— IBOGAINE —
Nature's Cure for Addiction

A treatment using ibogaine, a plant-derived alkaloid, has a such a high success rate in overcoming drug and alcohol dependence that it should be more widely available.

Compiled and Edited from various sources, including Howard S. Lotsof's Ibogaine internet web site: www.ibogaine.org/index.html

A plant-derived alkaloid, known as ibogamine, or, more commonly, ibogaine, has been shown to interrupt the vicious cycle of drug dependence, and has proven particularly effective in combatting addictions to opiate narcotics, such as heroin, as well as to cocaine, other stimulants and even alcohol. It has enormous promise in a treatment that can overcome both the physiological and the psychological problems of addiction, for not only does it speed up the withdrawal process but it also helps addicts identify the root cause of their addictions and regain control of their lives.

Ibogaine is primarily derived from the tropical plant *Tabernanthe iboga* which is native to equatorial western Africa, but it has also been isolated from the *Tabernanthe manii*, *Ervatamia yunnanensis* and *Ervatamia orientalis* shrubs, all being members of the Apocynacae family. Analysis of the leaves of *Ervatamia orientalis*, which grows mainly in the coastal monsoonal vine thickets north of Gordon Vale in far north Queensland, Australia, shows significant levels of ibogaine and an overall alkaloid profile that mirrors the tabernanthe plant, *Tabernanthe iboga*.

Tabernanthe is a plant traditionally used by tribes in the Congo and the Gabon regions of western Africa where it is valued for its variety of medicinal properties, as well as its inebriating, aphrodisiacal and hallucinogenic effects when taken in high doses for ceremonial purposes. Bwiti tribesmen would chew the tabernanthe root on their long hunting trips to reduce their fatigue levels and need for sleep. The Mitsogho Bwiti and Fang Bwiti tribes in the Gabon reportedly use the plant in their initiation rituals for both sexes. Ceremonial preparation would require higher-dose intake by prolonged chewing of tabernanthe leaves and root bark. The resultant experiential phases have been compared with the stages observed in a study of 150 people who reported a so-called "near-death experience" (NDE) while temporarily clinically dead.

The tabernanthe root was first taken out of Gabon in 1864 but was not described by the Museum of Natural History in Paris until 1889. A crystallised alkaloid was first isolated from the root by Dybowsky and Landrin in 1901 and named ibogaine. Several studies were undertaken by French pharmacologists in 1901-05, establishing ibogaine's clinical efficacy as a cardiac stimulant and as a treatment for neurasthenia and asthenia. However, it was not until 1939 that serious study was resumed into this alkaloid's pharmacodynamic and therapeutic potential. And because of its hallucinogenic properties, the tabernanthe root managed to find its way into modern-day drug culture in the 1960s, and was eventually classified as an hallucinogenic substance in 1972.

In 1962, a former heroin addict named Howard Lotsof took ibogaine in his search for a new way to get high. After a 36-hour hallucinogenic experience, he no longer craved heroin. Most remarkably, he did not experience any of the severe withdrawal symptoms that are normally associated with heroin. Lotsof shared the ibogaine with six other addicts, five of whom lost their desire for heroin. These amazing results prompted Lotsof to conduct further research into ibogaine's effects. From the mid-'80s to early '90s, Lotsof secured several patents on the use of ibogaine for treating drug and alcohol addiction.

Although about 40 addicts have been treated in the Netherlands since 1990 in controlled trials, ibogaine has not yet been approved by the US Food & Drug Administration (FDA) for use in the United States, where it is illegal.

MEDICAL TREATMENT OF CHEMICAL DRUG DEPENDENCE

Ibogaine is not a substitute for narcotics or stimulants; it is not addictive, and it is given in a single administration modality (SAM). It is a chemical dependence interrupter. Retreatment may occasionally be needed until the person being treated with ibogaine is able

to extinguish certain conditioned responses related to the drugs they abuse.

Early data suggest that, for many patients, a period of approximately two years of intermittent treatments may be required to attain the goal of long-term abstinence from narcotics and stimulants. The majority of patients treated with ibogaine remain free from chemical dependence for a period of three to six months after a single dose. Approximately 10 per cent of patients treated with ibogaine remain free of chemical dependence for two or more years from a single treatment, and an equal percentage return to drug use within two weeks after treatment. Multiple administrations of ibogaine over a period of time are generally more effective in extending periods of abstinence.

Ibogaine was first reported to be effective in interrupting opiate narcotic dependence disorders in US Patent No. 4,499,096 (Lotsof, 1985), cocaine dependence disorders in US Patent No. 4,587,243 (Lotsof, 1986), and poly-drug dependence disorders in US Patent No. 5,124,994 (Lotsof, 1992).

The initial studies demonstrating ibogaine's effects on cocaine and heroin dependence were accomplished in a series of focus group experiments by Howard S. Lotsof in 1962 and 1963.

Additional data on the clinical aspects of ibogaine in the treatment of chemical dependence were reported by Kaplan (1993), Sisko (1993), Sanchez-Ramos and Mash (1994), and Sheppard (1994).

Prior to ibogaine's evaluation for the interruption of various chemical dependencies, its use was reported in psychotherapy by Naranjo (1969, 1973), and at the First International Ibogaine Conference held in Paris (Zeff, 1987).

The use of ibogaine-containing plants has been reported for centuries in western Africa in both religious practice and in traditional medicine (Fernandez, 1982; Gollnhofer and Sillans, 1983, 1985).

An overview of the history of ibogaine research and use was published by Goutarel et al. (1993).

Claims of efficacy in treating dependencies to opiates, cocaine and alcohol in human subjects were supported in preclinical studies by researchers in the United States, the Netherlands and Canada. Dzoljic et al. (1988) were the first researchers to publish ibogaine's ability to attenuate narcotic withdrawal. Stanley D. Glick et al. (1992) at Albany Medical College published original research and a review of the field concerning the attenuation of narcotic withdrawal. Maisonneuve et al. (1991) determined the pharmacological interactions between ibogaine and morphine, and Glick et al. (1992) reported ibogaine's ability to reduce or interrupt morphine self-administration in the rat. Woods et al. (1990) found that ibogaine did not act as an opiate, and Aceto et al. (1991) established that ibogaine did not precipitate withdrawal signs or cause dependence.

Cappendijk and Dzoljic (1993) published ibogaine's effect in reducing cocaine self-administration in the rat. Broderick et al. (1992) first published ibogaine's ability to reverse cocaine-induced dopamine increases and, later, reported on ibogaine's reduction of cocaine-induced motor activity and other effects (1994). Research by Broderick et al. supported the findings of Sershen et al. (1992), that ibogaine reduced cocaine-induced motor stimulation in the mouse. Sershen (1993) also demonstrated that ibogaine reduced the consumption of cocaine in mice.

Glick (1992) and Cappendijk (1993) discovered in the animal model that multiple administrations of ibogaine over time were more effective than a single dose in interrupting or attenuating the self-administration of morphine and cocaine, supporting Lotsof's findings in human subjects (1985).

Popik et al. (1994) determined ibogaine to be a competitive inhibitor of MK-801 binding to the NMDA receptor complex. MK-801 has been shown to attenuate tolerance to opiates (Trujillo and Akil, 1991) and also to alcohol (Khanna et al., 1993). MK-801 has also shown a blockade of "reverse tolerance" of stimulants (Karler et al., 1989).

The *Tabernanthe iboga* plant, source of ibogaine.

Ibogaine's effects on dopamine (a substance hypothesised to be responsible for reinforcing pleasurable effects of drugs of abuse) and the dopamine system were found by Maisonneuve et al. (1991), Broderick et al. (1992) and Sershen et al. (1992). Ibogaine binding to the kappa opiate receptor was reported by Deecher et al. (1992).

Thus, a broad spectrum of mechanisms is evident by which ibogaine may moderate the use of substances as diverse as opiate narcotics, stimulants and alcohol.

PROGRESSION OF EFFECTS

Ibogaine's actions are understood to progress in three stages:

(1) A four-to-six-hour period emulating dreaming, in which intense sounds, lights, visual presentations and life-review thoughts or processes are experienced.

(2) A cognitive or intellectual period in which those experiences of the first phase are subjected to a high-energy evaluation process, then integrated into a new ego structure.

(3) A period of residual stimulation, eventually resulting in sleep. Lotsof describes this final phase as comprising three-to-four-hour sleep periods over a 24-to-40-hour duration. The subject rises from this in great form and with a new self-confidence.

By way of comparison, in a study of 150 people "rescued from death", American cardiologist Michael Sabom noted the following general progression as being typical of the near-death experience:

(1) Autoscopic phase: subjective feeling of being dead; peace and well-being; disembodiment; visions of material objects and events;

(2) Transcendental phase: tunnel or dark zone; evaluation of past life; light; access to a transcendental world/entering the light; encounters with other beings; return to life.

Interestingly, many of these individuals underwent a spectacular transformation during their NDEs. Many who reported seeing their whole lives flash before their eyes no longer feared death;

they felt stronger, more optimistic and calm, and were able to contemplate life more positively.

With ibogaine treatment, it is after the patient awakens that the effects are principally noticed, for the majority of patients no longer desire to seek or use the drugs they were abusing. However, it should be noted that the response to ibogaine is very individual, just as the patient has individual characteristics.

According to Lotsof, patients report a reduced need for sleep for a period of up to four months, sometimes for up to six months, after receiving the ibogaine treatment.

A young Dutch woman has written this about her experiences with ibogaine treatment:

"I lost interest in drugs in general because the effect of ibogaine goes far beyond their effect, though not necessarily in a pleasant way. Up until four months after the treatment I kept experiencing colours and light very intensely.

"Ibogaine was a mental process for me, a form of spiritual purification and a truth serum in which I had to experience its results through time. It's only now, after six months, that I can say I'm not addicted any more. It takes time to admit that there is no way back. Ibogaine is not a solution in itself, although it takes away withdrawal completely."

Ibogaine provides the means for overcoming addiction, but the subject must have the desire to change. Ibogaine helps the individual realise that all knowledge is available to cure him/herself, if that is truly what is desired.

The use of ibogaine-containing plants has been reported for centuries in western Africa in both religious practice and in traditional medicine.

CLINICAL PROTOCOLS

The effects of ibogaine treatment are viewed in three categories: acute, intermediate and long-term. The acute and intermediate effects have sometimes been referred to as the effects and after-effects. The two major effects of ibogaine are: (1) the ability to interrupt narcotic and stimulant withdrawal, and (2) the attenuation or elimination of the craving to continue to seek and use opiates, stimulants and alcohol (Lotsof, 1985, 1986, 1989).

Knowledge concerning the use of ibogaine in treating alcohol dependence is limited to: (1) a single-alcohol-only dependent patient, and (2) the attenuation and, in some cases, cessation of alcohol use in persons treated for poly-drug-dependence disorders.

Ibogaine's ability to treat nicotine dependence (Lotsof, 1991) has been seen in poly-drug-dependent subjects treated primarily for opiate and/or cocaine use.

Some general considerations should be noted regarding treatment protocols. The treatment team has four primary obligations:

(1) to earn the trust of the patient; (2) to maintain the comfort of the patient; (3) to assist the patient in interrupting his/her chemical dependence; and (4) to supply the psychosocial support network needed by the majority of patients to enable them to develop a sense of personal accomplishment and the ability to function as productive members of society. This is a process the Dutch treatment community refers to as "normalisation".

In the Lotsof Procedure™ (for which a manual is now being prepared), the sense of conflict seen in most treatment modalities between the doctor and patient over the immediate ceasing of drug use does not exist. If narcotic-dependent, the patient is allowed to continue the use of narcotics until a certain time prior to treatment with ibogaine. There is no conflict over opiate use before treatment because, as Lotsof maintains, ibogaine will either work to interrupt chemical dependence or it will not. In the procedure, any patient dependent on stimulants is not maintained on stimulants, and this practice has not created problems for patients or medical staff.

Before the Lotsof Procedure was conducted in hospitals under experimental trial conditions, addicted patients were allowed to use their personal supply of narcotics until the evening before ibogaine treatment. However, during hospital-administered ibogaine sessions, the narcotic-dependent patient is maintained on medications prescribed by the principal investigator during the three-to-five-day intake process preceding their treatment with ibogaine.

Even under these circumstances, patients' distrust of the medical establishment and their extreme fear of going into withdrawal has resulted in narcotics being smuggled into hospital environments. As a protective mechanism, all patients scheduled for ibogaine treatment in a hospital must undergo a thorough physical examination upon admission and allow all their possessions to be searched. This serves two important functions. Firstly, it limits the possibility of accidental overdose from secreted narcotics, stimulants or other drugs. Secondly, it allows a more comprehensive understanding of the patient's physical health, since many of the people seeking treatment for chemical dependence have often masked numerous health problems for years or even decades by self-medicating with illicit, addictive drugs.

ACUTE EFFECTS REGIMEN

The acute effects of ibogaine are dramatic. The initial reaction in the patient is usually noted within 45 minutes after intake of the oral dose, and full effects are generally evident within 2 to 2½ hours. The earliest subjective indication by the patient of ibogaine's effects is the report of a pervasive oscillating sound. The patient tends to lie down, and if asked, to stand or walk, shows signs of ataxia.

The protocol for the Lotsof Procedure stipulates that the patient remain in bed with as little movement as possible from the time of ibogaine administration, as nausea associated with ibogaine use has been proven to be motion-related or, in later stages (those longer than four hours after administration), possibly a psychosomatic reaction to previously repressed traumatic experiences.

In addition to keeping the patient as still as possible, the protocol suggests use of a non-phenothiazine anti-nauseant, as phenothiazines may interfere with the psychoactive properties of ibogaine. If the patient vomits in less than 2½ hours after the administration of ibogaine, an examination of the regurgitated material should be made to determine how much ibogaine may have already been absorbed by the patient. A rectal infusion of ibogaine to supplement the lost portion of the dose may be provided if it is not possible for this dose to be administered orally, but only if the patient has previously consented to this mode of dosing.

AUTONOMIC RESPONSES

During the first through the fifth hour of ibogaine treatment there is a moderate rise in the patient's blood pressure of 10 to 15 per cent and, in some cases, an associated decline in the pulse rate. The most significant autonomic changes occur between 1½ and 2½ hours after administration of therapeutic doses of ibogaine.

In many cases, the patient's pulse rate is elevated due to anxiety prior to the administration of ibogaine.

THE VISUALISATION PHASE

One of ibogaine's principal effects during its first phase of action is to produce a state which emulates dreaming, although the subject is fully awake and has the ability to respond to the treatment staff's questions.

In most cases, people under the influence of a therapeutic dose of ibogaine do not wish to speak. They prefer instead to pay close attention to the visual presentation of memories or phenomena they are experiencing—visuals that have been noted to have both Freudian and Jungian connotations.

The experiencing of visual material is rapid. Some patients have described it as a movie run at high speed; others as a slide show, each slide containing a motion picture of a specific event or circumstance in the viewer's life. In either case, the presentation of visual material is so compressed and fast-moving that distraction of the patient for even a moment may interfere with the process of abreaction. Therefore, medical staff intrusion should be kept to a minimum during the ibogaine treatment's primary phase.

COGNITIVE EVALUATION

Following the visualisation stage, the second phase of ibogaine's action during the Lotsof Procedure is one of the patient's intellectual evaluation of previous experiences and decisions. This occurs after the visualisation phase, which generally ends abruptly after a three-to-five-hour period. However, individual reactions and variations are the norm and not the exception within the parameters of the procedure.

As for the patient's own process of evaluation, when the patient made various decisions in the past, those decisions appeared to be the only options available at the time. However, with ibogaine's effect of allowing the re-evaluation of life, actions and behaviour, the patient can come to understand that alternatives to those original decisions were in fact available at the time. This knowledge appears to enable the patient to modify his/her current behaviour and cease dependence on drugs.

BEHAVIOURAL IMMOBILITY DURING TREATMENT

During the periods of visualisation, and extending into the stage of cognitive evaluation, patients will demonstrate a state of behavioural immobility (Depoortere, 1987) during which brainwave patterns associated with dreaming and sleep, but distinct from those states, are represented by rhythmic, slow activity of 4-6 Hz. These EEG patterns are associated with a state characterised by a lack of movement.

Some early observers of the Lotsof Procedure (Kaplan, 1990) initially believed that the condition represented paralysis, but when patients were asked to stand and move around, the patients were able to do so, albeit with some degree of difficulty.

The acute interruption of the craving to seek and use drugs of abuse is unique to the Lotsof Procedure as a treatment modality for chemical dependence disorders.

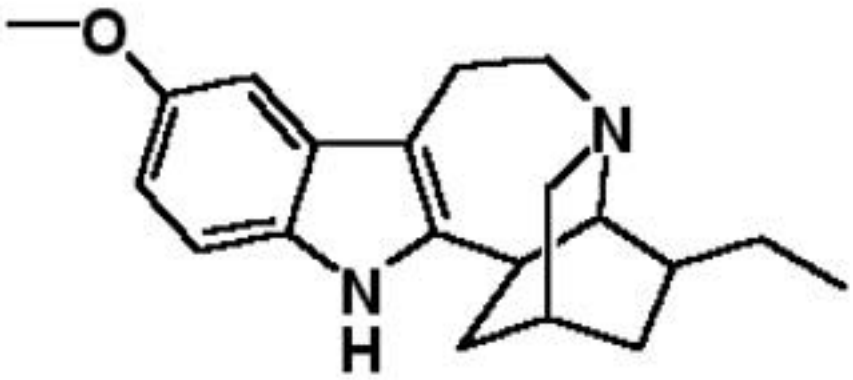
INTERRUPTION OF CRAVING

The acute interruption of the craving to seek and use drugs of abuse is unique to the Lotsof Procedure as a treatment modality for chemical dependence disorders. This effect is generally not noticed by the patient until ibogaine's principal actions (visualisation, cognitive evaluation, behavioural immobility and significant residual stimulation) are no longer evident and the patient has had the opportunity to sleep.

The initial recognition of lack of craving is usually noticed 48 to 72 hours after ibogaine administration. In a minority of treatments, recovery and absence of craving may be evident to the person being treated in as little as 24 hours. Medical staff, on the other hand, usually notes the absence of craving in the patient in 45 minutes to 1½ hours after ibogaine administration.

According to NDA International (the medical development company set up by Howard Lotsof to supervise experimental therapy according to the Lotsof Procedure), its experience gained in recent years with the treatment of 20 persons outside the US has shown that the majority of patients may need a series of treatments before they can extinguish their conditioned responses (cravings) to a long history of chemical dependence. However, three of these patients only required a single treatment to interrupt their chemical dependence for a minimum of two years.

One advantage of ibogaine is that it allows the patient time-periods free of craving. It is during those times that the



Molecular structure of ibogaine

psychiatrist, social worker, therapist, paraclinician and the patient often bond into a cohesive working group to accomplish a state of long-term non-dependence to the drug(s) of abuse for which the patient is under treatment.

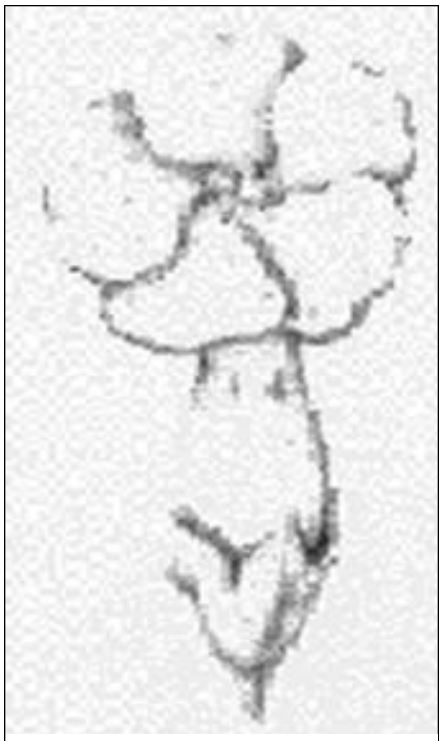
REDUCTION IN THE NEED FOR SLEEP

In all cases, ibogaine reduces the patient's need for sleep to as little as three or four hours a night. This effect may last a month or more, gradually returning to normal.

Two theories have been put forth concerning this effect. The first theory suggests the long-lasting bioavailability of ibogaine or one of its metabolites. This is in keeping with pharmacokinetic studies conducted at the University of Miami (Mash, 1995). The second theory suggests that the cause is the decrease in the psychological requirements for sleep associated with the need to dream. Evidence supporting this theory is that ibogaine promotes an intense emulation of dreaming that lasts for many hours during its acute stage of activity.

The reduction in the need for sleep is viewed by the majority of patients as a discomfort, since they have been used to using drugs and sleep as escape mechanisms. These patients may require some mild form of sedation during the first days after ibogaine treatment.

Normal precautions should be taken in providing sedatives to persons with a history of chemical dependence. In a minority of cases, patients have used this newly available time to advantage in their busy work schedules.



Flower of the *Tabernanthe iboga* plant.

PSYCHOSOCIAL SUPPORT

All aspects of treatment for chemical dependence disorders common to other treatment modalities are common to ibogaine treatment.

The patient's characteristics in terms of psychopathology, behaviour, societal accomplishments, as well as the skills of the treatment team are significant to treatment outcomes.

In cases when the patient already has the occupational and educational skills needed to succeed in society, the task may be somewhat easier.

In cases where the patient does not have those societal skills, or lacks medical care for disorders other than chemical dependence, care and training must be provided through psychosocial support structures.

Trauma suffered by the patient during childhood appears to play an important part in the drive for love and the fear of abandonment that is common to many of the patients treated (Bastiaans, 1991).

Many of the accepted parameters of distance between the therapist and the patient are not effective in ibogaine treatment. Patients will require closer and more intensive guidance, and generally be more open to it. They will require faster intervention to learn societal skills and to overcome and understand objectively the various traumas experienced during their lives.

Ibogaine is therefore not a treatment modality for clinicians whose preference is simply to administer a pill and then distance themselves from their patients.

References

1. Aceto, M.D., "Biological Evaluation of Compounds for Their Physical Dependence Potential and Abuse Liability", US National Institute for Drug Abuse (NIDA) Research Monograph 119(506):520-523, 1991.
2. Bastiaans J., "The Psychiatric and Psychosomatic Dimensions of Trauma", personal communication with Howard Lotsof, 1991.
3. Broderick, P.S., Phelan, F.T. and Berger S.P., "Ibogaine Alters Cocaine-Induced Biogenic and Psychostimulant Dysfunction, but Not [3H]GBR-12935 Binding to the Dopamine Transporter Protein", *Problems of Drug Dependence 1991*, Proceedings of the 53rd Annual Scientific Meeting, CPDD, NIDA Research Monograph 119:285, 1992.
4. Broderick, P.S., Phelan, F.T., Eng, F. and Wechsler, T., "Ibogaine Modulates Cocaine Responses which are Altered Due to Environmental Habituation: *In Vivo* Microvoltammetric and Behavioural Studies", *Pharmacology, Biochemistry and Behaviour* 49(3):711-728, 1994.
5. Cappendijk, S.L.T. and Dzoljic, M.R., "Inhibitory Effects of Ibogaine on Cocaine Self-Administration in Rats", *European Journal of Pharmacology* 241:261-265, 1993.
6. Deecher, D.C., Teitler, M., Soderlund, D.M., Bornmann, W.G., Kuehne, M.E. and Glick, S.D., "Mechanisms of Action of Ibogaine and Harmaline Congeners Based on Radioligand Binding Studies", *Brain Research* 571:242-247, 1992.
7. Depoortere, H., "Neocortical Rhythmic Slow Activity During Wakefulness and Paradoxical Sleep in Rats", *Neuropsychobiology* 18:160-168, 1987.
8. Dzoljic, E.D., Kaplan, C.D. and Dzoljic, M.R., "Effects of Ibogaine on Naloxone-Precipitated Withdrawal Syndrome in Chronic Morphine-Dependent Rats", *Archive of International Pharmacodynamics* 294:64-70, 1988.
9. Fernandez, J.W., *Bwiti: An Ethnography of Religious Imagination in Africa*, Princeton University Press, USA, 1982.
10. Glick, S.D., Rossman, K., Rao, N.C., Maisonneuve, I. M. and Carlson, J.N., "Effects of Ibogaine on Acute Signs of Morphine Withdrawal in Rats: Independence From Tremor", *Neuropharmacology* 31(5):497-500, 1992.
11. Glick, S.D., Rossman, K., Steindorf, S., Maisonneuve, I.M. and Carlson, J.N., "Effects and Aftereffects of Ibogaine on Morphine Self-Administration in Rats", *European Journal of Pharmacology* 195:341-345, 1992.
12. Gollnhofer, O. and Sillans, R., "L'Iboga Psychotrope Africain" ("Iboga, An African Psychotropic Agent"), *Psychotropes* 1(1):11-27, 1983.
13. Gollnhofer, O. and Sillans, R, "Usages Rituels de l'Iboga au Gabon" ("Ritual Uses of Iboga in Gabon"), *Psychotropes* 2(3):95-108, 1985.
14a. Goutarel, R., Gollnhofer, O. and Sillans, R., "Pharmacodynamics and Therapeutic Applications of Iboga and Ibogaine", *Psychedelic Monographs & Essays* 6:71-111, 1993.
14b. Goutarel, R., Gollnhofer, O. and Sillans, R., "L'Iboga et l'ibogaine contre la dépendance aux stupéfiants: Pharmacodynamie et applications psychothérapeutiques", *Psychotropes* 8(3), 1993.
15. Judd, B., personal communication with Howard Lotsof, 1993.
16. Kaplan, C.D., personal communication with Howard Lotsof, 1990.
17. Kaplan, C.D., Ketzer, E., de Jong, J. and de Vries, M., "Reaching a State of Wellness: Multistage Explorations in Social Neuroscience", *Social Neuroscience Bulletin* 6(1), Winter 1993.

Continued overleaf ...

FEMALE PATIENT SAFETY

One 24-year-old female patient, treated for chemical dependence in ibogaine trials in the Netherlands, died from undiagnosed causes. Although the autopsy did not determine the cause of her death, ibogaine levels of 0.75mg/litre were recorded in the blood. This level has not been seen to be toxic in animal research or in prior human studies undertaken by NDA International.

Subsequent to this death (and to a previously reported death of a Swiss woman who received ibogaine during a psychotherapy session in Europe which was totally unrelated to NDA's research program) the FDA excluded women from the clinical trials presently taking place at the University of Miami.

However, the FDA decision is contrary to the gender guidelines of the National Institutes of Health. The NIH guidelines call for the inclusion of women at the earliest stages of clinical trials, in order to provide the greatest determination of safety for women.

NDA International reports that 30 per cent of its patients have been women and they have shown no negative effects from taking ibogaine either during or after treatment. However, considering all of the circumstances, the Lotsof Procedure should be administered only in a hospital or clinic with the patient under continuous staff observation and electronic monitoring.

An ongoing international research program is collating evidence to determine an hypothesis for the cause of death of the woman in the Netherlands. Additionally, NDA International is seeking Swiss Government cooperation concerning the death of the Swiss woman.

IBOGAINE TREATMENT LOGISTICS

NDA International, Inc. has received many requests for information concerning ibogaine treatment for drug dependence.

Currently, the only treatments authorised by a health ministry are administered in hospitals in Central America as part of an experimental program.

Due to the significant expense of hospitalisation and the logistics involved with this program, the base cost per person can be US$15,500 for participation in treatment within a group of three. The cost of an individually scheduled treatment is significantly higher. These charges do not include the preliminary medical evaluations, or transportation to and costs in New York for required pre-treatment and post-treatment medical screening.

Start campaigning to make Ibogaine available in Australia now! Contact your local politician and health-care practitioner and help get loved ones off drugs of dependency!

If you require further information on referral for ibogaine treatment, write to:

Intake Department
NDA International, Inc.
PO Box 100506
Staten Island, NY 10310-0506
USA

Your letter should provide statements including the following:

1. You are seeking treatment with ibogaine.

2. You understand that ibogaine is an experimental therapy and has not been approved by the US Food and Drug Administration (FDA). If you reside in a country other than the United States, state that you understand that ibogaine is an experimental therapy and not approved by the health department of the country where you reside.

3. Include a brief history of your current and previous drug use.

4. List any medical conditions you are aware of, and any medications you are taking or have taken during the last 12 months (including, but not limited to, anti-psychotics, anti-depressants, cardiovascular drugs, anti-hypertensives and anti-convulsants).

5. Print and sign your complete name and provide your mailing address, telephone number and e-mail address. ∞

References (continued)

18. Karler, R, Calder, L.D., Chaudhry, I.A., Turkanis, S.A., "Blockade of 'Reverse Tolerance' to Cocaine and Amphetamine by MK-801", *Life Sciences* 45:599-606, 1989.
19. Khanna, J.M., Kalant, H., Shah, G. and Chau, A., "Effect of D-cycloserine on Rapid Tolerance to Ethanol", *Pharmacology, Biochemistry and Behaviour* 45(4):983-986, 1993.
20. Lotsof, H.S., "Rapid Method for Interrupting the Narcotic Addiction Syndrome", US Patent No. 4,499,096, 1985.
21. Lotsof, H.S., "Rapid Method for Interrupting the Cocaine and Amphetamine Abuse Syndrome", US Patent No. 4,587,243, 1986.
22. Lotsof, H.S., "Rapid Method for Attenuating the Alcohol Dependency Syndrome", US Patent No. 4,857,523, 1989.
23. Lotsof, H.S., "Rapid Method for Interrupting or Attenuating the Nicotine/Tobacco Dependency Syndrome", US Patent No. 5,026,697, 1991.
24. Lotsof, H.S., "Rapid Method for Interrupting or Attenuating Poly-drug Dependency Syndromes", US Patent No. 5,124,994, 1992.
25. Maisonneuve, I.M., Keller, R.W., Jr, and Glick, S.D., "Interactions between Ibogaine, a Potential Anti-Addictive Agent, and Morphine: An *In Vivo* Microdialysis Study", *European Journal of Pharmacology* 199:35-42, 1991.
26. Mash, D.C., Douyon, R., Hearn, W.L., Sambol, N.C. and Sanchez-Ramos, J., "A Preliminary Report on the Safety and Pharmacokinetics of Ibogaine", *Biological Psychiatry*, 1995 (in press).
27. Naranjo, C., "Psychotherapeutic Possibilities of New Fantasy Enhancing Drugs", *Clinical Toxicology* 2(2):209, 1969.
28. Naranjo, C., *The Healing Journey*, Pantheon Books/Random House, New York, USA, 1973, pp. 174-228.
29. Personal communication with Howard Lotsof (author wishes to remain anonymous), 1994.
30. Popik, P., Layer, R.T. and Skolnick, P., "The Putative Anti-addictive Drug Ibogaine is a Competitive Inhibitor of [3H]MK-801 Binding to the NMDA Receptor Complex", *Psychopharmacology* 114:672-674, 1994.
31. Sabom, M.B., *Recollections of Death*, Harper & Row, USA, 1982.
32. Sanchez-Ramos, J. and Mash, D.C., "Ibogaine Research Update: Phase I Human Study", *MAPS*, IV(4):11, Spring 1994.
33. Sershen, H., Hashim, A., Harsing, L. and Lajtha, A., "Ibogaine Antagonizes Cocaine-Induced Locomotor Activity in Mice", *Life Sciences* 50:1079-1086, 1992.
34. Sershen, H., Hashim, A. and Lajtha, A., "Ibogaine Reduces Preferences for Cocaine Consumption in C57BL/6By Mice", *Pharmacology, Biochemistry and Behaviour* 47(1):13-19, 1994.
35. Sheppard, S.G., "A Preliminary Investigation of Ibogaine: Case Reports and Recommendations for Further Study", *J. Substance Abuse Treatment* 11(4):379-385, 1994.
36. Sisko, B., "Interrupting Drug Dependency with Ibogaine: A Summary of Four Case Histories", *MAPS* IV(2):15-23, Summer 1993.
37. Trujillo, K.A. and Akil, H., "Inhibition of Morphine Tolerance and Dependence by NMDA Receptor Antagonist MK-801", *Science* 2512:85-87, 1991.
38. *Village Beat*, New York, NY, May 1990.
39. Woods, H.W., Medzihardsky, F., Smith, C.B., Winger, G.D. and Prince, C.P., *1989 Annual Report: Evaluation of New Compounds for Opioid Activity*, NIDA Research Monograph 95(563):655-656, 1990.
40. Zeff, L., First International Ibogaine Conference, Paris, France, January 1987 (videotape).